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Autoimmune Thrombocytopenia and Hepatitis C Virus Infection

To the Editor: Hepatitis C virus (HCV) infection can be associated with several autoimmune manifestations [1,2]. An association between autoimmune thrombocytopenia (AITP) and HCV infection has been suggested in two case reports [3,4]. However, the association between HCV infection and AITP has not been confirmed.

We examined the prevalence of serum anti-HCV antibodies in 44 patients with AITP (idiopathic and secondary), diagnosed between January 1994–August 1995. Anti-HCV was detected by third-generation enzyme immunoassay and confirmed with recombinant immunoblot assay (RIBA-3) in 4 of 44 patients (9%).

The anti-HCV-positive patients, 4 women with a mean age of 53 years (range, 32–73 years), had an isolated thrombocytopenia of >6 months' duration, with normal bone-marrow aspiration, and absence of splenomegaly and of any underlying disorder other than HCV infection (cryoglobulin test and HBsAg were negative). There was history of blood transfusion in 1 patient, with no known risk factor for HCV in the other patients. Serum alanine aminotransferase (ALT) was increased in 2 patients for >6 months, but was normal in the other 2 patients. The mean platelet count at diagnosis was 13.2×10^9/l (range, $9–20 \times 10^9$/l). Liver biopsy was not performed because of thrombocytopenia. Patients were treated initially with prednisone (1 mg/kg/day for 3 weeks). Response was achieved in the 4 patients, but they relapsed after supression of prednisone. Liver function tests were unchanged during and after steroid treatment. Two of 3 patients responded to intravenous immunoglobulin (400 mg/kg/day for 5 days). One patient had undergone a splenectomy with a complete response, but she relapsed 2 months later.

Thrombocytopenia in the presence of chronic liver disease is classically attributed to hypersplenism. However, our finding of a 9% prevalence of HCV antibodies among patients with AITP, and of the association of HCV infection with various autoimmune disorders, supports the hypothesis that an immune mechanism can mediate thrombocytopenia in some patients with chronic hepatitis C. Further studies are needed to establish the incidence of AITP in HCV infection.

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Pelvic Osteomyelitis in a Sickle-Cell Patient Receiving Deferoxamine

To the Editor: Deferoxamine (DFO), an effective and relatively safe chelating agent used for iron reduction in chronically transfused patients, has several recognized side effects but has never been associated with osteomyelitis. We report here on the case of a patient with sickle-cell anemia (SCA) receiving DFO who developed osteomyelitis of the left ilium. The site of infection was directly under the location of previous DFO subcutaneous infusions, suggesting a local, rather than systemic, complication.

A 17-year-old male with SCA presented with a 9-day history of left hip pain and fever. He had been transfused regularly over the previous 6 years to prevent recurrent cerebrovascular accidents, and had been using DFO subcutaneously 3 nights a week over the previous 2 years. He exclusively used the buttocks and surrounding soft tissue for subcutaneous infusions. The area of pain corresponded to the site of deferoxamine infusions. Radiographs of the pelvis and chest were unremarkable, but magnetic resonance imaging (MRI) of the pelvis showed increased signal over the lateral aspect of the left iliac wing and surrounding soft tissue, consistent with a soft-tissue infection and concurrent osteomyelitis. Blood culture was positive for *Staphylococcus aureus* within the first 24 hr of hospitalization, obviating the need for biopsy. He made a full recovery with antibiotic therapy, and has no evidence of recurrence 22 months later.

Osteomyelitis is a well-documented complication in patients with SCA. The most common sites of osteomyelitis in patients with SCA are the long bones, particularly of the lower extremities. The ilium is a rare site of osteomyelitis [1]. In patients with SCA and other foci of infection, osteomyelitis in the pelvis may be subclinical and perhaps underdiagnosed [2]. Osteomyelitis, regardless of location, may be difficult to distinguish from acute bone infarction in SCA, even with the use of MRI [3]. Culture of an organism from blood or bone can confirm the diagnosis.

The relationship between iron overload, chelation therapy, and infection is not clear. In vitro data suggest that patients with iron overload receiving chelation therapy with DFO may have an increased risk of bacterial infection [4]. In our patient, it is likely that the infection was not caused by the DFO itself, but rather from bacterial contamination that occurred during preparation or infusion of the drug or insertion of the needle. *Staphylococcus aureus* is known to be often present as part of the normal human flora, and foreign bodies are predisposing risk factors for infection [5]. The needle itself may have come into direct contact with the bone, or it may have provided a portal of entry, causing an initial cellulitis with secondary osteomyelitis. Hematogenously spread osteomyelitis from another site seems unlikely, given the location of the lesion (under the injection site) and the absence of clinical or radiographic lesions elsewhere.

We conclude that osteomyelitis may complicate subcutaneous DFO infusions. When administering subcutaneous DFO infusions, meticulous attention must be given to aseptic technique, and bony prominences should be avoided.

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